

## CLAIMS

1. An inactive  $\text{Ca}^{2+}$ /calmodulin-dependent protein kinase II $\alpha$  (CaMKII $\alpha$ ) knockin nonhuman animal, wherein a CaMKII $\alpha$  gene of one or both of homologous chromosomes is substituted into an inactive type so that an inactive CaMKII $\alpha$  is expressed, and thereby a protein kinase activity of the CaMKII $\alpha$  is specifically impaired while a calmodulin binding capacity of the CaMKII $\alpha$  and a capacity of multimerizing subunits are maintained.
2. The inactive CaMKII $\alpha$  knockin nonhuman animal according to claim 1, wherein the inactive CaMKII $\alpha$  knockin nonhuman animal is produced by a gene targeting method.
3. The inactive CaMKII $\alpha$  knockin nonhuman animal according to claim 1 or 2, wherein one or a plurality of amino acid residues in a catalytic domain of the CaMKII $\alpha$  has been modified.
4. The inactive CaMKII $\alpha$  knockin nonhuman animal according to claim 3, wherein one or a plurality of amino acid residues that is required for binding to ATP has been modified.
5. The inactive CaMKII $\alpha$  knockin nonhuman animal according to claim 4, wherein a lysine residue that is required for binding to ATP has been modified.
6. The inactive CaMKII $\alpha$  knockin nonhuman animal according to any one of claims 1 to 5, wherein the inactive CaMKII $\alpha$  knockin nonhuman animal is a rodent animal.
7. The inactive CaMKII $\alpha$  knockin nonhuman animal according to claim 6, wherein the inactive CaMKII $\alpha$  knockin nonhuman animal is a mouse.
8. The inactive CaMKII $\alpha$  knockin nonhuman animal according to claim 1, wherein brain's nucleus accumbens has lower neuronal activity as compared to that of a wild type.

9. An inactive  $\text{Ca}^{2+}$ /calmodulin-dependent protein kinase II $\alpha$  (CaMKII $\alpha$ ) knockin cell, wherein a CaMKII $\alpha$  gene of one or both of homologous chromosomes is substituted into an inactive type so that an inactive CaMKII $\alpha$  is expressed, and thereby a protein kinase activity of the CaMKII $\alpha$  is specifically impaired while a calmodulin-binding capacity of the CaMKII $\alpha$  and a capacity of multimerizing subunits are maintained.